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Acute Brachial Artery Thrombosis as the Initial Manifestation of Human Immunodeficiency Virus Infection

Misha Witz,[1] Jonathan Lehmann,[1] and Ze'ev Korzets[2]*

[1]Unit of Vascular Surgery, Meir Hospital, Kfar-Saba, and the Sackler School of Medicine, University of Tel-Aviv, Tel-Aviv, Israel
[2]Department of Nephrology, Meir Hospital, Kfar-Saba, and the Sackler School of Medicine, University of Tel-Aviv, Tel-Aviv, Israel

Thrombosis of upper extremity arteries is most commonly due to atherosclerosis of the proximal subclavian artery, trauma, or catheter-related injury. In the absence of an identifiable cause, a search for a hypercoagulable state is indicated. Hematologic manifestations of human immunodeficiency virus (HIV) infection and AIDS are frequent occurrences (Coyle TE. Med Clin N Am 1997;81:449–476). The most important of these are cytopenias (anemia, neutropenia, and thrombocytopenia). The incidence and severity of cytopenia are generally correlated to the stage of the HIV infection. In addition, various coagulation abnormalities have been reported in HIV-infected patients. Apart from thrombocytopenia, these have included a prolonged APTT due to the presence of lupus anticoagulant, an increased prevalence of protein S and heparin cofactor II deficiency, and hypoalbuminemia-related fibrin polymerization defects (Toulon P. Ann Bio Clin (Paris) 1998;56:153–160). HIV infection has also been associated with endothelial dysfunction. Although for the most part asymptomatic, elevated D-dimer levels have been found in HIV-infected patients, suggesting the existence of a prethrombotic state. In fact, clinical thrombosis eventuates in 2% of these patients (Toulon, 1988). Documented thromboses have involved both veins and arteries. We hereby present a patient who developed an acute thrombosis of his brachial artery as the initial manifestation of HIV infection. Am. J. Hematol. 64:137–139, 2000. © 2000 Wiley-Liss, Inc.

Key words: brachial artery; thrombosis; HIV infection

CASE REPORT

This 53-year-old heterosexual male was admitted because of painful paresthesias of his right forearm which had suddenly began 5 days previously. He had a history of recurrent primary syphilis for which penicillin had been administered. He was a heavy smoker (35 pack years). On examination, cyanosis of all fingers of the right hand, which was colder than the left, was evident. Although painful, he retained normal movement of his fingers. The brachial and radial pulses were not palpable. Blood pressure (left arm) was 120/70 mmHg. Other extremities appeared normal. An echodoppler examination revealed a low-resistance flow pattern in the brachial and ulnar arteries. Hemoglobin was 11.7 g/dL, white cell count 7,500/μL, platelets 205,000/μL, albumin 3.7 g/dL, globulin 4.3 g/dL, PT 92%, INR 1.1, PTT 27 sec. Total protein S was 17 mg/L (normal 13–21), protein C activity 78% (70–130), and anti-thrombin III 96% (80–120). Anticardiolipin antibodies were positive: GPL 55 (<23), MPL 29 units/mL (<11).

A right subclavian arteriogram showed complete occlusion of the brachial artery (Fig. 1). The patient underwent thrombectomy of the right brachial artery. No evidence of atherosclerosis was noted. After the operation, both brachial and radial pulses were palpable. He was,

Abbreviations: APTT, activated partial thromboplastin time; HIV, human immunodeficiency virus; AIDS, acquired immunodeficiency syndrome; CD4, CD4+ T lymphocytes; GPL, IgG antiphospholipid antibody; MPL, IgM antiphospholipid antibody.

*Correspondence to: Dr. Ze'ev Korzets, Department of Nephrology, Meir Hospital, Kfar-Saba, Israel, 44281.

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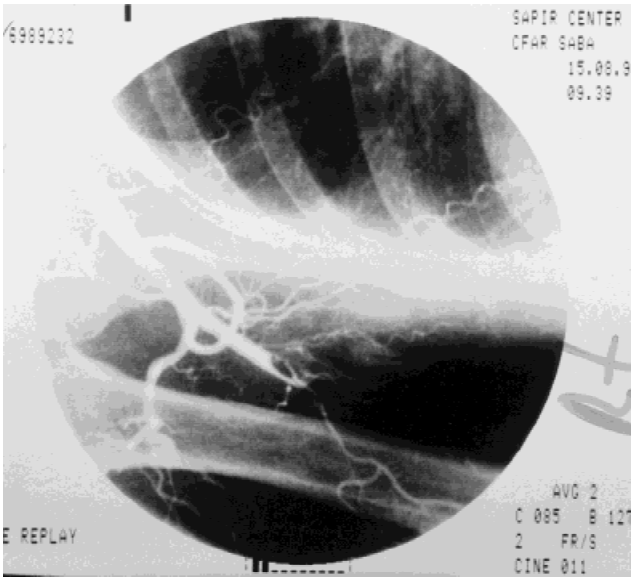

Fig. 1. Subclavian arteriogram showing complete occlusion of the right brachial artery.

thereafter, maintained on heparin and discharged a month postoperatively for further outpatient evaluation, on warfarin therapy.

An echocardiogram showed no thrombus, normal valves and no patent foramen ovale. During the ensuing two months, the patient complained of increasing fatigue and weight loss. Erythrocyte sedimentation rate was 100 mm (Westergren). Antinuclear antibody was negative. Complement (C3 and C4) levels were within normal limits. Quantitative immunoglobulins were IgG 2,789, IgA 750, and IgM 390 mg/dL. HIV was positive with a CD4 count of 112.

Treatment with zidovudine and cotrimoxazole was initiated. Eight months postoperatively he was admitted with fever and a confusional state. Extensive investigation revealed no apparent cause. At the time, his radial pulse was not palpable, the ulnar only barely. He had minimal wrist and finger flexor function. He continued to waste and died 2 months later.

DISCUSSION

Immunologically mediated diseases, whether autoimmune or immune deficient, have a propensity for thrombosis. Among the diseases demonstrating this thrombotic tendency are, notably, primary antiphospholipid antibody syndrome, systemic lupus erythematosus, immune thrombocytopenic purpura or its variant thrombotic thrombocytopenic purpura, and immunohemolytic anemia [3]. Clinical thrombosis has been reported to occur in 2% of HIV-infected patients [2]. The pathogenesis of the increased risk of thrombosis in this population is, as yet, not completely understood but is, probably, of a multifactorial nature.

The prevalence of lupus anticoagulant in HIV infection has ranged as high as 70% [2]. Anticardiolipin antibodies have been reported in 46–90% of HIV-infected patients [1]. In this setting, they have rarely been associated with multiple transient ischemic attacks and stroke [4], avascular necrosis of bone [5], and skin necrosis [6]. Sugerman et al. have recently reported that acquired protein S deficiency is common in HIV-infected children (75%), significantly more prevalent in those with a CD4 count <200 [7]. Another physiological coagulation inhibitor, heparin cofactor II, has also been reported to be deficient in HIV infection. Other possible contributing factors are hypoalbuminemia-related fibrin polymerization defects, endothelial dysfunction and abnormalities of the fibrinolytic system [2]. Narayanan et al. described multiple abdominal venous thromboses and splenic hematoma in a patient seropositive for HIV [8]. No cause for the hypercoagulable state was detected in this patient. Celiac artery thrombosis, resulting in splenic infarction and pancreatitis in another HIV-infected patient, was reported by Aouad et al. [9].

Idiopathic thrombosis of arteries of the upper extremity is an exceptionally rare occurrence. A literature review has yielded only 4 such cases [10–12]. Arterial thrombosis involving the upper limbs, therefore, mandates a thorough coagulation screen and close follow up. Our patient is a case in point. Having presented with brachial artery thrombosis, anticardiolipin antibodies were found on this admission. However, HIV was only diagnosed 3 months later. To the best of our knowledge, this is the first reported case of brachial artery thrombosis in a HIV-infected patient with anticardiolipin antibodies. Furthermore, the arterial thrombosis proved to be the initial manifestation of this patient's HIV infection.

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